Extracorporeal Shock Wave Lithotripsy of Urinary Calculi
Theory, Efficacy, and Adverse Effects

JAMES M. KELLEY, *Tucson*

This paper was chosen as the winner of THE WESTERN JOURNAL OF MEDICINE's 1990 Medical Student Writing Competition. The contest was open to all students attending medical schools in the West and will be repeated annually.

LINDA HAWES CLEVER, MD
Editor

Extracorporeal shock wave lithotripsy (ESWL) for the treatment of upper urinary tract stone disease is held in high regard by the public and the profession. Although the efficacy is good (77.4% to 100%) for the treatment of 1- to 2-cm stones in select patients, ESWL may require the assistance of adjuvant procedures in as many as 26% of patients and may need repeating in as many as 32% of patients. These represent more difficult situations in which larger, more numerous, or harder stones may be present and in which ureteral stones are manipulated before treatment. The predominant adverse effect of ESWL treatment is the microvascular disruption of the tissues through which the shock waves pass. In addition, the procedure is painful, with many patients requiring narcotic analgesia. Long-term complications such as the new onset of hypertension have occurred in as many as 8% of treated patients, but much speculation about the long-term effects remains.

(Kelley JM: Extracorporeal shock wave lithotripsy of urinary calculi—Theory, efficacy, and adverse effects. West J Med 1990 Jul; 153:65-69)

Often in medicine, the benefits gained from a therapeutic intervention are weighed against the risks of carrying out that intervention. A simple balance between gains and losses, or present effects and future results, exists that teeters between both outcomes. Such may be the case with extracorporeal shock wave lithotripsy (ESWL).

Since its first clinical application in 1980, ESWL's technologic progress has advanced at a sprinter's pace. Currently more than 300 extracorporeal lithotripters are used in 32 countries worldwide and more than 500,000 people have received shock wave treatment of renal or ureteral stones.[1,2] Despite this clinical experience, fundamental questions still remain about the nature of shock wave energy, its effects on the tissues through which it passes, the proper dosage, and the absolute role of ESWL in the treatment of stone disease.

With the procedure embraced by both the public and the profession, the fervor to develop new, less costly, and safer devices for delivering shock waves has made keeping track of the players and the styles of play difficult. The Dornier company, from which pioneering lithotripsy studies were generated and the producer of the first clinically available extracorporeal shock wave lithotripter, the HM-1, has found itself among an ever-increasing pack of rookie companies trying to capture a portion of the growing market. With the increased competition and the rapidly expanding body of knowledge, new variants of extracorporeal shock wave-generating devices have been emerging almost yearly. As an example of just how quickly things are progressing, since its development of the HM-1 nearly a decade ago, the Dornier company has produced the HM-2, HM-3, HM-4, MPL 9000, and MFL 5000 models.[2]

With the newer, second-generation lithotripters, attempts have been made either to improve on the original design or to develop novel approaches to the task of stone fragmentation. The changes made in various lithotripter components include variations in the energy source, focusing devices, coupling medium, and stone localization systems.[2]

Shock Wave Principles

Although highly theoretical, current thinking regarding shock wave behavior holds that, in the spark-gap type of shock wave generator, the sudden jump of charge between two electrodes immersed in deionized water results in the instantaneous evaporation of water around the electrodes. This sudden evaporation creates an expanding gas bubble that compresses the surrounding water medium and produces an increase in the adjacent water density.[3] Resisting further compression, the water medium allows transmission of the propagated pressure wave at an increased velocity. This change in velocity results in the characteristic shock wave appearance of a sharp pressure rise followed by a gradual decay.[4]

Because of a stone's ability to effectively resist compression, it is thought that tensile forces cause the characteristic peeling off of outer layers that has been observed to occur with fragmentation. The tensile forces are essentially the result of reflected and refracted shock waves, which develop an area of low pressure behind them as they journey away from the area of contact. These tensile forces are thought to stress the stone's intrinsic ability to resist longitudinal stretching, resulting in the formation of fractures from the outermost surface inward.[4] In the spark-gap type of lithotripter machines, the strength of the administered shock waves can generally be varied by changing the voltage (about 18 to 24 kV) across the spark-generating electrode.[4]

The author is a third-year medical student at the University of Arizona College of Medicine, Tucson.
Reprint requests to James M. Kelley, 504 W LaMar Rd, Phoenix, AZ 85013.

ABBREVIATIONS USED IN TEXT

ESWL = extracorporeal shock wave lithotripsy
NAG = β-*N*-acetyl-D-glucosaminase

Cellular Effects of Shock Waves

At the cellular level, investigation with high-energy shock waves has been mainly in the study of their potential for use against neoplastic cell lines. These studies, however, could serve partly as an analysis of their effects at the cellular level.

In a study using rat prostate carcinoma cells and human melanoma cell lines in vitro, Russo and co-workers observed a selective diminution of cells in the G2 and M phases of the cell cycle following exposure to 800 to 1,500 shock waves. The complementing in vivo study showed a delay in tumor growth among cells treated directly in an animal host or treated before injection into an animal host.[5] The finding of a shock wave-induced antiproliferative effect has been supported by the work of others, but the selective cell phase diminution was not observed.[6]

Similarly, Randazzo and associates examined indices of cell viability, growth, cell attachment, and electron-microscopic appearance in renal carcinoma cells versus normal human embryonic kidney cells[7] in vitro. The renal carcinoma cells were treated with 1,400 or 2,000 shock waves at 18 kV, causing a notable decrease in viability, growth, and attachment over what was observed in the normal human embryonic kidney cells. Electron microscopy showed substantial damage to both cell types with improvement seen in both at 120 hours when given the lower shock wave dose, but favoring the embryonic kidney cells morphologically.[7] In vivo experimentation using FANFT, a carcinogen-induced mouse bladder tumor transplanted into the hind legs of C3H/He mice and exposed to 1,400 shock waves, showed an inhibitory effect on tumor growth at this dose.[7]

Showing obvious cellular effects, these study results also raise the question of cell vulnerability at particular cell cycle phases. They suggest also that some cells, when damaged, may have the capability to undergo repair after exposure to shock waves.

Concerning normal cell lines, pioneering work by Chaussy demonstrated no in vitro change in leukocyte number or function following shock wave exposure.[8] In a study by Yang and associates, however, a significant cytotoxic effect was observed in bone marrow cells exposed in vitro. This effect was observed to be dose dependent with increasing shock wave exposure: As few as 500 shock waves at 18 kV decreased cell viability by 50%. In addition, colony-forming ability was reduced following exposure to shock waves.[9]

Admonishing the cautious interpretation of tumor cell studies, Laudone and colleagues showed that the air-fluid interface present in containment vessels used for in vitro studies significantly enhanced the cytotoxic effects of administered shock waves. They noted, too, that shock wave effects differ substantially for cells in suspension versus cells in tissue, making comparisons difficult. Furthermore, in trying to reproduce the antiproliferative effects observed by other investigators, using the implantation of AT3 tumor cells, they witnessed no significant change in tumor doubling times versus controls after treatment of the tumors with 1,000 shock waves at 24 kV. They concluded from the latter study, however, that the choice of tumor type may have been inappropriate. The AT3 type is homogenous, lacking the surface areas of a more variegated tumor.[10]

Tissue Effects: Animal Studies

Preliminary studies showed that shock waves pass through living tissue with little attenuation in wave energy.[8] Work with animal models and in vitro systems suggested that shock waves produce only minimal degrees of tissue injury that resolves quickly. An early study by Chaussy involving the surgical deposition of a calculus into the renal pelvis of 17 dogs with subsequent fragmentation showed successful fragmentation and fragment clearance in 13 of the animals, each given 500 shock waves, without evidence of tissue injury or loss of renal function.[8] Notable exceptions, however, were the massive alveolar and vascular rupture produced in directly exposed rat lung and, separately, hemolysis of erythrocytes exposed in vitro to shock waves.[8] The former led to the practice of shielding the lung during treatment, and the latter was attributed to the presence of aged erythrocytes in the test blood specimen.

Allowing gross and histologic examination within and adjacent to the focus of maximum shock wave energy, continued studies in animals have played a necessary part in assessing the effects of high-energy shock waves on living tissues. Animal subjects used to date include rats, rabbits, dogs, and pigs.

In studies of rats, Recker and co-workers showed by scanning electron microscopy of treated kidneys the diffuse loss of microvilli and ciliae on the cell surface of tubules, cell vacuolization, desquamation, and sporadic glomerular rupture.[11] In studies of the ovary, an organ likely to receive shock waves during the treatment of distal ureteral calculi, no important effects on follicle number or fertility were noted in female rats with exposure to 1,500 focused shock waves at 15 kV.[12] Given the possibility of unintentional vertebral shock wave exposure in children undergoing ESWL, the proximal tibia of 5-week-old rats was submitted to 1,500 shock waves at 20 kV, and the animals were killed two, four, and ten weeks after treatment. Focal growth plate dysplasia was found in 8 (44%) of the 18 rats when compared with age-matched controls, with 2 (33%) of the 6 ten-week animals showing extensive, rather than focal, dysplasia.[13]

For a general assessment of acute and chronic effects of ESWL on various tissues, Graff and associates, using the rabbit model, observed posttreatment bleeding and ulceration of the exposed rectum, intermuscular and intramuscular hematomas, and subperiosteal bleeding at 48 hours. Chronic effects, apparent at one to two months, included aseptic marrow necrosis, damage to osteocytes, and evidence of bone remodeling.[14] Study of the morphologic and functional effects of 3,000 shock waves (at 20 kV) on the rabbit kidney showed the acute (seven days) changes of focal subcapsular hemorrhage, tubular dilatation, and interstitial hemorrhage; chronically (two months), only focal interstitial fibrosis was seen. When both kidneys were assessed for kidney functional losses, the only significant findings were a transient four-day decrease in creatinine clearance and a transient three-day increased concentration of urinary free hemoglobin, both of which returned to baseline.[15]

Several studies have examined the direct effects of shock waves on the kidney, using the canine model. In contradistinction to the early findings of Chaussy, Brendel showed renal hemorrhages (as much as 0.5 cm in size), interstitial edema with interstitial widening, and renal capsule petechiae following shock wave exposure to the canine kidney.[16(p141)] To study the acute and chronic effects on the kidney and try to correlate shock wave dose with degree of

injury, Newman and colleagues studied six kidneys of mongrel dogs. The acute-effects group comprised four kidneys, each having exposure to a different shock wave dosage (1,776, 4,500, 6,000, and 8,000 shocks), and the animals were killed at 48 to 72 hours. Corticomedullary hemorrhages were found universally in the kidneys of this group, especially involving thin-walled vessels. Three of four kidneys (75%) also showed perirenal hemorrhage or hematoma. The chronic-effects group—two kidneys exposed to 1,600 or 8,000 shock waves and the animals killed 30 days after treatment—showed evidence of fibrosis, which was smaller in the less heavily treated kidney. Surprisingly, neither kidney in the acute or chronic group exposed to 8,000 shock waves showed complete necrosis. All kidneys, whatever dose given, sustained modest injury.[17] The unusually high shock wave dosages given in this experiment were for assessing extremes in dosage and effects.

Using more conventional shock wave dosages, other studies have examined shock wave exposure to kidneys for acute and chronic changes.[18-20] After 1 to 24 hours, macroscopic changes include perirenal fat hemorrhages, a relative increase in treated kidney size, subcapsular hematomas, overlying muscle hemorrhages, and hemorrhages of the renal parenchyma, capsule, or hilum.[18-20] On histologic examination there were focal and diffuse interstitial hemorrhage, venous thrombosis involving arcuate and interlobular veins, and tubular dilatation.[18-20] Chronic changes (seven days to three months) were not observed macroscopically.[19,20] On histologic examination, chronic renal changes included only focal areas of streaky fibrosis and calcium deposition.[19,20] By electron microscopy, the microvascular wall was the site of the greatest injury.[18] When assessing shock wave dose effects, injuries were more serious above 500 shocks, but no difference was appreciable when comparing the effects of 1,500 with 3,000 shock waves.[18]

Pig model studies, too, have shown renal structural disruption with exposure to high-energy shock waves. Thinking pigs to be a better model for human kidney than other model kidneys, Muschter and co-workers showed acute renal injury consisting of perirenal and intrarenal hematomas, renal parenchymal damage, and parenchymal necrosis following renal parenchymal ESWL treatment.[21] Chronic changes were demonstrated by Begun and Laswon, who examined pig kidneys four weeks after treatment.[22] They found focal scarring in the treated kidney circumscribed by marginally vascularized areas of injury in which interstitial and perivascular fibrosis, partial glomerular hyalinization, and tubular injury were present, resembling end-stage renal disease.[22] The mechanism(s) of shock wave-induced tissue injury has not yet been elucidated. Under consideration have been the compressive and tensile forces of the wave, thermal events, free radical effects, or a cavitation phenomenon.

Direct forces are a plausible explanation for such injury in biologic structures having different impedances, but thermal injury is unlikely.[23] Free radicals produced by high-energy shock waves may play a role in cytotoxicity. They have, however, shown little effect on a target cell assay when compared with those of cobalt 60 origin.[24] A cavitation phenomenon seems the most widely accepted explanation for observed tissue effects.[3,5,23] Sometimes called pseudocavitation, it is the product of small gas or vapor bubbles by rapid pressure changes in a liquid.[25] A momentary decrease in pressure behind an advancing shock wave is thought to cause local vaporization within a liquid, or undissolution of a dissolved gas, producing tiny bubbles. Such bubbles have been observed in in vitro experiments. It is the motion of these bubbles that could cause tissue injury.[23,25]

Clinical Experience

Reports of clinical success with ESWL abound in the literature. Since its first clinical application nearly a decade ago, widespread acceptance has provided the opportunity to review the experience in a sizable population of treated patients.

Regarding efficacy, early studies by Chaussy and associates, in which 945 patients were treated over a three-year period, showed that 89.5% of treated patients were stone free at three months' follow-up.[26] In the United States Cooperative Study of Extracorporeal Shock Wave Lithotripsy, follow-up of 739 treated cases at three months showed freedom from stones and all detectable residual fragments in 77.4% of cases.[27] Overall, the estimated efficacy of ESWL ranges from 38% to 100%. Some of this variation is attributable to the treatment of difficult and large stones, a significant number of patient retreatments, and the assistance of adjuvant procedures.

It has been suggested that a single kidney stone with a largest diameter of 2.0 cm or as many as three stones of less than or equal to 1.0 cm provide an excellent target for ESWL.[28] Such was supported in the US cooperative study in which the model size of treated stones was 1 to 2 cm.[27] In dealing with large stones, Roth and Beckman found that only 38% of patients with calculi larger than 3.0 cm are treated successfully by ESWL monotherapy.[29] In the US cooperative study, most patients required a single ESWL treatment, but one, two, and more treatments were required in 16%, 14%, and 2% of patients, respectively.[27]

The use of percutaneous or endoscopic procedures as an adjuvant to ESWL therapy is commonplace. In some studies, these procedures have been used routinely in as many as 26% of patients to reposition a stone, remove an excessive fragment burden following treatment, or allow calyceal drainage of urine.[30,31] Overall success rates, too, have improved with adjuvant therapy. In one study, treatment using ESWL alone was 80% successful, climbing to 90% at three months' follow-up with the addition of adjuvant measures.[32] In many cases, the stone burden is the key factor in determining whether assistance is necessary, as the increased number of fragments released from a large (>3.0 cm) stone or numerous stones raises the risk of urinary tract obstruction.[31] In addition, the variable success of in situ ureteral stone fragmentation has been improved by the pretreatment manipulation of ureteral stones into a more advantageous position in the renal pelvis.[33-35]

Adverse Effects

Complications seen routinely with ESWL treatment of stone disease are cutaneous petechiae or subcutaneous hematomas at the shock wave entry and exit sites (especially in thin patients) and costovertebral angle or lower quadrant pain, with as many as 40% of these patients requiring narcotics for pain relief.[36] Gross hematuria is observed immediately in virtually all patients and typically resolves by 24 to 48 hours after treatment.[3,36,37] Obstruction of urinary flow by fragments also occurs, with a general frequency of between 6% and 25%.[33,38,39] The piling of stone fragments in the ureter, called steinstrasse, has been described throughout the literature and in one study occurred in 5% of 600 patients.[32] After large or infected stones are fragmented, infection can develop in an obstructed calyceal system, resulting in septicemia. Such complications have occurred in less than 5% of the cases examined.[32,38,40]

Other complications include pneumonitis in 1.2% of patients,[27] prolonged ileus in 10%,[32] and transient pyrexia in 30% of patients.[32] Perirenal hematomas, occurring in as many as 2.5% of patients in general following ESWL treatment, have been found with greater frequency (as many as 3.8%) in patients with poorly controlled hypertension at the time of treatment.[41]

Morphologically, computed tomography and magnetic resonance imaging have shown significant changes in the posttreatment kidney. Computed tomographic studies acutely reveal a general relative increase in the size of the treated kidney (9% in one study), subcapsular hematomas, intrarenal hemorrhages, small subcapsular fluid collections of an unknown type, and perirenal fascial thickening.[42] Magnetic resonance imaging findings concur; in the examination of 39 kidneys in one study, 29 (74%) showed one or more morphologic changes.[43] In addition, focal or diffuse loss of the corticomedullary junction occurred in 16 (41%) of those 39 patients.[43] Other investigators have observed similar changes.[3,39]

In the investigation for changes in renal function, various methods have been employed. Enzyme studies by Kishimoto and co-workers and Assimos and colleagues have revealed transient elevations in renal tubular enzymes for as long as seven days after treatment.[44,45] Kishimoto and associates observed shock wave dose-related increases in the levels of cell-escaped lactate dehydrogenase, aspartate aminotransferase, and free hemoglobin lasting as long as four days in serial blood specimens and an increasing β-*N*-acetyl-D-glucosaminase (NAG) and creatinine clearance trend through the completion of the observation period (day 4).[44] Assimos and co-workers noted an increase in the urine concentrations of the proximal tubule and brush border enzymes NAG, γ-glutamyltransferase, and β-galactosidase, with peak values reached at day 3 and returning to baseline beginning at five to seven days.[45] Marcellan and Servio also observed increases in serum lactate dehydrogenase and urine NAG levels, but these levels remained within the upper limits of the normal range.[46] Similarly, Schulze and associates measured a fourfold increase in the level of kidney epithelial antigens, using monoclonal antibodies, by the fourth post-ESWL treatment day.[47] These studies suggest that nephron injury does occur, but whether the decrease in some values indicates repair of this injury or progression to a nonfunctional state, without further leakage, is unclear.

Another study by Gilbert and colleagues assessing post-ESWL renal function more traditionally found nephrotic-range proteinuria for three to six months after treatment, but no significant change in the calculated glomerular filtration rate, fractional sodium excretion, or urine osmolality among treated patients.[48] These findings suggest that altered permeability of the glomerular basement membrane occurs with shock wave exposure.

Flow studies, too, have shown changes in effective renal plasma flow following ESWL treatment. Using quantitative radionucleotide renography scans, Kaude and co-workers found a 5% difference in the effective renal plasma flow before and after treatment in 10 of 33 patients.[39] Thomas and colleagues also found a significant difference in the effective renal plasma flow between treated and untreated sides, when greater than 1,600 shock waves were applied to the treated kidney.[49]

With the exception of the flow studies and the rabbit studies of Gunasekaran and associates,[15] these studies have the inherent limitation of being unable to selectively study just the treated kidney. A study by Kulb and co-workers involving patients with solitary kidneys showed a significant posttreatment rise in serum creatinine levels above 177 μmol per liter (2 mg per dl) in only 3 of 68 patients.[50] Given the vast nephron reserve in the kidney, however, a serious injury might not be appreciable simply by examining serum creatinine levels.

Concern over the long-term complications of ESWL treatment has recently been raised. Newly diagnosed hypertension has been found in treated patients. At 6 to 12 months after treatment, Lingeman and Kulb noted among 95 patients an 8% incidence of new-onset hypertension requiring drug therapy and a 15% incidence of new diastolic pressure elevations not requiring therapy.[51] Similar observations were made in two other studies by Newman and associates and Williams and colleagues, each reporting an increased incidence of new-onset hypertension in 8% of 148 and 91 patients, respectively. In both studies, these newly hypertensive patients required treatment.[52,53] In addition, at 17 to 21 months of follow-up, the Williams study also reported a substantial decrease in the effective renal plasma flow of the treated kidney in 21 of 148 treated patients.[53]

Conclusion

As Lingeman and co-workers put it,

> The rapid acceptance and adoption of ESWL has been facilitated by the false perception that this technology is entirely safe and that shock wave treatment does not induce severe, acute or chronic side effects.[37(p797)]

Although the overall mortality associated with this procedure is low (0.02% in a survey of 62,793 patients),[54] the effects of this energy on living tissue require a need for its judicious use.

Of added concern is the problem of repeat stone formation, with the frequency of stone recurrence found in one study to be 50% to 70% of patients,[55] who may undergo repeated ESWL treatments. Such retreatment would be expected to take its toll on a treated kidney. Will long-term accumulated injuries cause premature aging of treated kidneys, resulting in a decreased parenchymal reserve in later years? Although little long-term evidence exists for permanently altered renal function, the substantial nephron reserve of the kidney could conceivably compensate for sizable parenchymal injuries such that these injuries will not be manifested immediately. Effective at fulfilling its purpose, some ESWL models, however, still produce sufficient discomfort in treated patients to require narcotic analgesia. An added factor is that a substantial number of these patients will require additional procedures, endoscopic or invasive, indicating that ESWL alone is not a cure for all stone problems and requires the optimal selection of patients.

REFERENCES

1. Drach GW: Shock-wave lithotripsy: How much is enough?, *In* Walker VR, Sutton AL (Eds): Urolithiasis. New York, Plenum, 1989, pp 911-915

2. Chaussy CG, Fuchs GJ: Current state and future developments of noninvasive treatment of human urinary stones with extracorporeal shock wave lithotripsy. J Urol 1989; 141:782-789

3. Brendel W: Shock waves: A new physical principle in medicine. Eur Surg Res 1986; 18:177-180

4. Hunter PT II: The physics and geometry pertinent to ESWL, chap 2, *In* Riehle RA Jr (Eds): Principles of Extracorporeal Shock Wave Lithotripsy. New York, Churchill Livingstone, 1987, pp 13-27

5. Russo P, Stephenson RA, Mies C, et al: High energy shock waves suppress tumor growth in vitro and in vivo. J Urol 1986; 135:626-628

6. Oosterhof GON, Smits GAHJ, de Ruyter JE: The in vitro effect of electromagnetically generated shock waves (Lithostar) on the Dunning R3327 PAT-2 rat prostatic cancer cell-line—A potentiating effect on the in vitro cytotoxicity of vinblastin. Urol Res 1989; 17:13-19

7. Randazzo RF, Chaussy CG, Fuchs GJ, et al: The in vitro and in vivo effects of extracorporeal shock waves on malignant cells. Urol Res 1988; 16:419-426

8. Chaussy C: Extracorporeal Shock Wave Lithotripsy. New York, Karger, 1986

9. Yang C, Heston WD, Gulati HS, et al: The effect of high energy shock waves (HESW) on human bone marrow. Urol Res 1988; 16:427-429

10. Laudone VP, Morgan TR, Huryk RF, et al: Cytotoxicity of high energy shock waves: Methodologic considerations. J Urol 1989; 141:965-968

11. Recker F, Ruben H, Hofstadter F, et al: Ultramorphological Acute and Long-term Lesions of ESWL in rat kidney. Sixth World Congress on Endourology and Extracorporeal Shock Wave Lithotripsy, Paris, 1988

12. McCullough DL, Yeaman LD, Bo WJ, et al: Effect of shock waves on the rat ovary. J Urol 1989; 141:670-674

13. Yeaman LD, Jerome CP, McCullough DL: Effects of shock waves on the structure and growth of the immature rat epiphysis. J Urol 1989; 141:670-674

14. Graff J, Pastor J, Richter KD: Effect of High Energy Shock Waves on Body Tissue (Abstr). Proceedings of the Fifth World Congress on Endourology and Extracorporeal Shock Wave Lithotripsy, Cairo, 1987, p 260

15. Gunasekaran S, Donovan JM, Chvapil M, et al: Effects of extracorporeal shock wave lithotripsy on the structure and function of the rabbit kidney. J Urol 1989; 141:1250-1254

16. Brendel W: Effect of shock waves on canine kidneys, *In* Gravenstein JS, Peter K (Eds): Extracorporeal Shock-Wave Lithotripsy for Renal Stone Disease: Technical and Clinical Aspects. Stoneham, Mass, Butterworth, 1986

17. Newman R, Hackett R, Senior D, et al: Pathological effects of ESWL on canine renal tissue. Urology 1987; 29:194-200

18. Delius M, Enders G, Xuan ZR, et al: Biological effects of shock waves: Kidney damage by shock waves in dogs—Dose dependence. Ultrasound Med Biol 1988; 14:117-122

19. Jaeger P, Redha F, Uhlschmid G, et al: Morphological changes in canine kidneys following extracorporeal shock wave treatment. J Endourol 1988; 2:205-213

20. Jaeger P, Redha F, Uhlschmid G, et al: Morphological changes in canine kidneys following extracorporeal shock wave treatment. Urol Res 1988; 16:161-166

21. Muschter R, Schmeller NT, Scheu W, et al: ESWL and Renal Damage: An Experimental Study Using the Modified Dornier Lithotripter HM-3 (Abstr). Proceedings of the Fifth World Congress on Endourology and Extracorporeal Shock-Wave Lithotripsy. Cairo, 1987, p 233

22. Begun FP, Laswon RK: Renal Injury From Focused Electrohydraulic Shock Waves. Sixth World Congress on Endourology and Extracorporeal Shock Wave Lithotripsy, Paris, 1988

23. Delius M: This month in investigative urology: Effect of extracorporeal shock waves on the kidney (Editorial). J Urol 1988; 140:390

24. Morgan TR, Laudone VP, Heston WDW, et al: Free radical production by high energy shock waves—Comparison with ionizing radiation. J Urol 1988; 139:186-189

25. Fischer N, Muller HM, Gulhan A, et al: Cavitation effects: Possible cause of tissue injury during extracorporeal shock wave lithotripsy. J Endourol 1978; 2:215-220

26. Chaussy C, Schmiedt E, Jocham D, et al: Extracorporeal shock-wave lithotripsy (ESWL) for treatment of urolithiasis. Urology (special issue) 1984; 23:59-66

27. Drach GW, Dretler S, Fair W, et al: Report of the United States Cooperative Study of Extracorporeal Shock Wave Lithotripsy. J Urol 1986; 135:1127-1133

28. Pettersson B, Tiselius HG: One year follow-up of an unselected group of renal stone formers treated with extracorporeal shock wave lithotripsy. J Endourol 1989; 3:19-30

29. Roth RA, Beckman CF: Complications of extracorporeal shock wave lithotripsy and percutaneous nephrolithotomy. Urol Clin 1988; 15:155-166

30. Fuchs G, Miller K, Rassweiler J, et al: Extracorporeal shock wave lithotripsy: One year experience with the Dornier lithotripter. Eur Urol 1985; 11:145-149

31. Alken P, Hardeman S, Wilbert D, et al: Extracorporeal shock wave lithotripsy (ESWL): Alternatives and adjuvant procedures. World J Urol 1985; 3:48-52

32. Coptcoat MJ, Webb DR, Kellett MJ, et al: The complications of extracorporeal shockwave lithotripsy: Management and prevention. Br J Urol 1986; 58:578-580

33. Chaussy C, Schmiedt E: Shock wave treatment for stones in the upper urinary tract. Urol Clin 1983; 10:743-750

34. Mueller SC, Wilkert D, Thueroff JW, et al: Extracorporeal shock wave lithotripsy of ureteral stones: Clinical experience and experimental findings. J Urol 1986; 135:831-834

35. Lingeman JE, Shirrell WL, Newman DM, et al: Management of upper ureteral calculi with extracorporeal shock wave lithotripsy. J Urol 1987; 138:720-723

36. Newman RC, Bezirdjian L, Steinbock G, et al: Complications of extracorporeal shock wave lithotripsy: Prevention and treatment. Semin Urol 1986; 4:170-174

37. Lingeman JE, Woods J, Toth PD, et al: The role of lithotripsy and its side effects. J Urol 1989; 141:793-797

38. Sofras F, Karayannis A, Kostakopoulos A, et al: Methodology, results and complications in 2000 extracorporeal shock wave lithotripsy procedures. Br J Urol 1988; 61:9-13

39. Kaude JV, Williams CM, Miller MR, et al: Renal morphology and function immediately after extracorporeal shock-wave lithotripsy. AJR 1985; 145:305-313

40. Lingeman JE, Newman RC, Mertz JHO, et al: Extracorporeal shock wave lithotripsy: The Methodist Hospital of Indiana experience. J Urol 1986; 135:1134-1137

41. Knapp PM, Kulb TB, Lingeman JE, et al: Extracorporeal shock wave lithotripsy-induced perirenal hematomas. J Urol 1988; 139:700-703

42. Rubin JI, Arger PH, Pollack HM, et al: Kidney changes after extracorporeal shock wave lithotripsy: CT evaluation. Radiology 1987; 162:21-24

43. Baumgartner BR, Dickey KW, Ambrose SS, et al: Kidney changes after extracorporeal shock wave lithotripsy: Appearance on MR imaging. Radiology 1987; 163:531-534

44. Kishimoto T, Yamamoto K, Sugimoto T, et al: Side effects of extracorporeal shock-wave exposure in patients treated by extracorporeal shock-wave lithotripsy for upper urinary tract stones. Eur Urol 986; 12:308-313

45. Assimos DG, Boyce WH, Furr EG, et al: Urinary enzyme levels after extracorporeal shock wave lithotripsy (ESWL) (Abstr). J Urol 1986; 137:143A

46. Marcellan FJR, Servio LI: Evaluation of renal damage in extracorporeal lithotripsy by shock waves. Eur Urol 1986; 12:73-75

47. Schulze H, Falkenberg FW, Mondorf AW, et al: Enhanced excretion of kidney-derived antigens in the urine of patients after ESWL treatment. J Urol 1986; 137:323A

48. Gilbert BR, Riehle RA, Vaughan EP Jr: Extracorporeal shock wave lithotripsy and its effect on renal function. J Urol 1988; 139:482-485

49. Thomas R, Roberts J, Sloane B, et al: Effect of extracorporeal shock wave lithotripsy on renal function. J Endourol 1988; 2:141-144

50. Kulb TB, Lingeman JE, Coury TA, et al: Extracorporeal shock wave lithotripsy in patients with a solitary kidney. J Urol 1986; 136:786-789

51. Lingeman JE, Kulb TB: Hypertension following extracorporeal shock wave lithotripsy (Abstr). J Urol 1987; 137:45A

52. Newman RC, Williams CM, Kaude J, et al: Hypertension Following Extracorporeal Shock Wave Lithotripsy. Fifth World Congress on Endourology and ESWL, Cairo, 1987

53. Williams CM, Kaude JV, Newman RC, et al: Extracorporeal shock-wave lithotripsy: Long-term complications. AJR 1988; 150:311-315

54. Ad Hoc Lithotripsy Committee Report, DL McCullough, Chair. Baltimore, American Urological Association, 1987

55. Sutherland JW: Recurrence following operations for upper urinary tract stones. Br J Urol 1954; 26:22